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Review

A current review on the regulation of dormancy in vegetative buds

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In this review, we examine current techniques and recent advances directed toward understanding cellular mechanisms involved in controlling dormancy in vegetative propagules. Vegetative propagules (including stems, rhizomes, tubers, bulbs, stolons, creeping roots, etc.) contain axillary and adventitious buds capable of producing new stems/branches under permissive environments. Axillary and adventitious buds are distinct in that axillary buds are formed in the axil of leaves and are responsible for production of lateral shoots (branches). Adventitious buds refer to buds that arise on the plant at places (stems, roots, or leaves) other than leaf axils. Both axillary and adventitious buds generally undergo periods of dormancy. Dormancy has been described as a temporary suspension of visible growth of any plant structure containing a meristem (Lang et al. 1987). Dormancy can be subdivided into three categories: (1) ecodormancy-arrest is under the control of external environmental factors; (2) paradormancy-arrest is under the control of external physiological factors within the plant; and (3) endodormancy-arrest is under the control of internal physiological factors. One common feature in all of these processes is prevention of growth under conditions where growth should otherwise continue. There is growing evidence that lack of growth is due to blockage of cell division resulting from interactions between the signaling pathways controlling dormancy and those controlling the cell cycle.

Nomenclature: Abscisic acid, ABA; CDK-activating kinase, CAK; cyclin-dependent protein kinase, CDK; extracellular-signal-regulated kinase, ERK; gibberellic acid, GA; growth factor receptor, GFR; mitogen-activated protein kinase, MAPK; underground adventitious buds, UABs; nuclear export signal, NES; nuclear localization signal, NLS; retinoblastoma, RB; virus-induced gene silencing, VIGS.

Key words: Leafy spurge, *Euphorbia esula* L., EPHES; cell cycle, signal transduction, perennial weeds.

Considerable effort has gone into the study of dormancy mechanisms in crop species. Because of their economic importance, dormancy has been extensively studied in potato (*Solanum tuberosum* L.) tubers (Suttle 2000) and in axillary buds of perennial woody plants such as apple (*Pyrus malus* L.) and populus (*Populus nigra* L.) (Crabbé and Barnola 1996; Pétel and Gendraud 1996; Stafstrom 1995). It is known that potato tuber meristems are endodormant when harvested but can be kept in a state of ecodormancy by cold storage after endodormancy is broken. Populus, a woody perennial, sets axillary buds in the fall. These buds acclimate and develop endodormancy, a mechanism that enables buds to resist freezing and dehydration stress in the winter. In addition to commercial species, many perennial weeds such as leafy spurge, field bindweed (*Convolvulus arvensis* L.), and Canada thistle [*Cirsium arvense* (L.) Scop.] undergo dormancy in a slightly different manner. Perennial weeds with creeping roots have adventitious buds on the root and crown (the junction of roots and stems) of the plants. These buds are collectively called "underground adventitious buds" (UABs). UABs of leafy spurge develop throughout the active growing season (Coupland et al. 1955) and are maintained in a growth-arrested state of paradormancy unless the aerial portion of the plant is killed (by frost, tillage, or herbicide application). However, in the fall, UABs of leafy spurge enlarge and appear to develop endodormancy but eventually shift to a state of ecodormancy for over-wintering (Schimming and Messersmith 1988). In this manner, UABs serve as the primary mechanism for perennial growth of such weeds. Dormancy, as a survival mechanism, makes leafy spurge a persistent problem in the United States and Canada, and current control measures have not proven very effective for controlling this weed.

Traditional studies on dormancy-related mechanisms have mainly focused on hormone changes (i.e., abscisic acid [ABA], auxin, cytokinin, gibberellic acid [GA], and ethylene) along with environmental factors and photosynthesis (Nooden and Weber 1978; Suttle 2000). The molecular nature and cellular basis of signals that carry out the processes of regulating dormancy are largely unknown. Identification of various important genetic components involved in root bud dormancy and growth is a way to approach more efficient and effective manipulation of such weeds. Recent advances in plant genetics and genomics have provided assorted tools to investigate the molecular mechanisms that control dormancy. Various approaches, such as mapping genes that influence dormancy using quantitative trait loci (QTL) analysis (Frewen et al. 2000; Paterson et al. 1995),

creating mutant lines to identify genes involved in dormancy (Napoli et al. 1999), differential display (Horvath and Olson 1998), and microarray (Anderson and Horvath 2000), have been used to investigate genes associated with dormancy. The objective of this review is to provide current findings on the regulation of dormancy in vegetative buds in perennial species. Since substantial progress has been achieved on morphogenesis of axillary buds from annual species like *Arabidopsis thaliana* L. and pea (*Pisum satvum* L.), some important discoveries from these species will also be included.

Physiology of Bud Dormancy

There is a substantial body of work that describes the basic physiology of dormancy in apical buds, a good bit less that addresses dormancy in axillary buds, and, until recently, virtually nothing was known about the control of dormancy in adventitious buds. Much of the work on apical bud dormancy has come from work on fruit trees. Early studies (completed in the past century) focused on environmental factors that controlled dormancy (Nooden and Weber 1978). Environmental factors, including day length, temperature, water, and nutrient availability, play major roles in control of ecodormancy and establishment of endodormancy in apical buds (Nooden and Weber 1978). For paradormancy, it was learned that actively growing apical buds could prevent growth of axillary buds below the apical meristem and that the subtending leaves of axillary buds had some effect on the growth of their axillary buds as well (Cline 1991; Zieslin and Halevy 1976). This paradormant phenomenon is also known as correlative inhibition. Excellent work based on these and other observations led to the discovery of plant hormones such as auxin, cytokinin, ABA, and GA (Nooden and Weber 1978). All of these hormones are implicated in control of all three types of dormancy (Nooden and Weber 1978). Additional experiments have found that sugars and nitrogen (Chao et al. 2000; Perry 1971), water potential (Borchert 1991), light quality and quantity (Nooden and Weber 1978), and developmental state (Nissen and Foley 1987) have direct effects on the levels and activity of these hormones in apical, axillary, and adventitious buds.

An important observation is that day length can affect development of endodormancy in all types of buds to some degree (Nooden and Weber 1978). In the Northern Hemisphere, short day length has been shown to induce dormancy in terminal buds of deciduous trees, apical buds of perennial and annual herbs, and adventitious buds of perennial herbs (Galitz 1994; Nooden and Weber 1978). In initial studies on this phenomenon, a compound was isolated from birch (*Betula* spp.) buds exposed to short days that, when exogenously applied, were able to induce dormancy-like characteristics in buds of seedlings (Eagles and Wareing 1963). This compound was later found to be ABA (Eagles and Wareing 1964). Additional evidence for a role of ABA in dormancy comes from genetic analysis of segregation of dormancy phenotypes in trees. It has been shown that the ratio of day to night needed to induce dormancy is progressively smaller in the same species collected at progressively higher latitudes (Pauley and Perry 1954). Mapping of genes involved in determining the sensitivity to day length identified loci with genes controlling ABA and phytochrome responsiveness (Frewen et al. 2000). Considerable work has shown that ABA can play a major role in maintenance of dormancy (Nooden and Weber 1978). In fact, there is unequivocal evidence that ABA can, by itself, induce dormancy in potato (Suttle and Hultstrand 1994). Given that variation in day length can influence the onset of dormancy in terminal buds of deciduous trees and that the ratio of red/far-red light increases in direct proportion to shortening day length, it is not surprising that phytochrome action has been implicated in this response (Nooden and Weber 1978). Phytochrome is known to be responsive to relative amounts of red and far-red light and is implicated in a number of different plant responses, including seed dormancy, changes in plant architecture under shaded or crowded growth conditions, and flowering (Neff et al. 2000). Among the genes shown to be responsive to phytochrome, two are known to play key roles in GA synthesis. GA biosynthetic genes encoding GA_{20} oxidase and 3β-hydroxylase have been shown to be controlled, at least in part, by phytochrome B (Jackson et al. 2000; Toyomasu et al. 1998; Yamaguchi et al. 1998b). GA sensitivity is also affected in several phytochrome B mutants of pea, cucumber (*Cucumis sativus* L.), and *Arabidopsis,* indicating that GA signaling is also altered by phytochrome (Lopez-Juez et al. 1995; Reed et al. 1996; Weller et al. 1994).

Environment plays a significant role in both the onset and maintenance of dormancy (ecodormancy). Cold temperatures can inhibit bud break and growth of adventitious and axillary buds under otherwise conducive conditions (Nooden and Weber 1978). Likewise, drought can also enhance dormancy and inhibit growth of buds under conditions that would otherwise permit growth (Borchert 1991). Both low temperature and low water potentials have been shown to increase ABA content in many plants (Chen et al. 1983). Thus, it appears that these observations also imply a role for ABA in dormancy of vegetative propagules.

Paradormancy (correlative inhibition) in axillary and adventitious buds is likely maintained primarily by factors produced in the growing meristems and young expanding leaves (see below). However, light has been shown to play a role in the regulation of growth in these buds as well (Horvath 1999; Zieslin and Halevy 1976). A compound produced by mature, photosynthesizing leaves has been shown to play a significant role in inhibition of adventitious bud growth in leafy spurge (Horvath 1998). This leaf-derived factor appears to be separate from the auxenic signal produced by meristematic regions (Horvath 1999). Also, this leaf-derived factor requires light and carbon fixation for its production and transport and may be sugar. In fact, root bud growth can be inhibited when plants are grown hydroponically in solutions containing sugar (Chao et al. 2000). Interestingly, exogenous application of GA is capable of overcoming the effect of the leaf-derived signal in leafy spurge (Horvath 1999). GA inhibitors can prevent adventitious bud growth under conditions that normally result in shoot development (W. S. Chao, unpublished results). GA has long been known to be antagonistic to ABA, and recent studies have indicated that sugars may act as an inhibitory signal to GA production and responsiveness (Perata et al. 1997).

As described above, environmental conditions such as light and temperature have been shown to act through various plant regulatory compounds (ABA, GA, and phyto-

chrome). In addition to these compounds, the hormones auxin and cytokinin have been implicated in dormancy control of adventitious and axillary buds (Cline 1991; Nissen and Foley 1987; Nooden and Weber 1978). Auxin is almost certainly the signal produced by the expanding meristem that directly or (more likely) indirectly is responsible for preventing growth of more distal axillary and adventitious buds (Cline 1991). In leafy spurge, application of exogenous auxin to isolated root sections reduces UAB growth (Horvath 1998; Nissen and Foley 1987). Also, auxin transport inhibitors can induce UAB growth in leafy spurge provided that the leaf-derived signal is absent (Horvath 1999). The manner in which auxin exerts this effect is unknown, and it is paradoxical that buds, whose growth is inhibited by exogenous auxin, actually produce auxin as soon as they are released from dormancy and resume growth (Stafstrom 1995). Elevated cytokinin levels have been implicated in breaking dormancy in adventitious and axillary buds (Stafstrom 1995). Both of these hormones have been shown to play an essential role in the control of growth and cell division in plants (Leyser et al. 1993; Soni et al. 1995). Ethylene is another plant hormone that has been implicated in control of dormancy in plants. Addition of an ethylene response inhibitor increased the rate of precocious sprouting in potato microtubers (Suttle 1998).

Molecular Mechanisms of Hormone Action in Bud Dormancy

Since the discovery that various plant hormones could influence the onset and maintenance of dormancy in various plant buds, there has been considerable effort to understand how these hormones act to control dormancy. Much work has focused on cloning genes that are differentially expressed in response to hormonal or environmental signals known to affect dormancy. Sequences in the promoter and transcribed regions of these genes that control transcription, RNA stability, RNA transport, or other aspects of gene expression have been identified. These sequences have been used to identify and clone genes encoding the proteins that bind to these sequences and cause their differential expression. Also, the controlling sequences of these differentially expressed genes were used to drive reporter genes, the expression of which could be easily monitored. Such "reporter gene constructs" have been successfully used to screen for mutations that affect the signaling pathways controlling their regulation. For example, studies have shown that auxin increases cell growth and division by interacting with the degradation pathways controlling the level of cyclins and other key components of the cell division machinery (Leyser et al. 1993). However, the process by which auxin inhibits growth of distal buds remains unknown. Recently, experiments using mutants with altered apical dominance have identified a couple of genes, *Rmsl1* and *Rmsl2,* whose products are transported through the plant and which interact with auxin to increase apical dominance (Beveridge et al. 2000).

Also, it is now known that ABA likely acts through the cyclin-dependant kinase inhibitor *Ick1* to prevent cell division (Figure 1) and to maintain dormancy in affected tissues (Wang et al. 1998). Recently it has been shown that a number of sugar-insensitive mutants that affect growth and photomorphogenesis were allelic to known ABA-insensitive mutants (Huijser et al. 2000; Laby et al. 2000). Combined, these results indicate a mechanism for control of adventitious bud growth by sugar that has been observed in leafy spurge. The induction of *Ick1* by ABA may also explain the inhibition of bud growth caused by low temperatures.

Less is known about the action of GA, but it is clear that GA can promote cell cycle activity in some way (Sauter et al. 1995). GA has been shown to be sufficient for induction of endoreduplication of DNA in *Arabidopsis* (Gendreau et al. 1999) and for induction of S-phase, but not M-phase, of the cell cycle in leafy spurge (Horvath et al., in preparation). It is possible that antagonism between GA and ABA signaling results in GA inhibiting *Ick1* production and thus allows cell cycle progression through the S-phase.

Work is just beginning to show how components of the cytokinin response pathways affect dormancy in adventitious buds. Recent work has indicated that overexpression of cytokinin biosynthetic genes increased cytokinin levels and reduced correlative inhibition in axillary buds (Faiss et al. 1997). Increased cytokinin levels can induce *Knat1,* a gene involved in meristem growth and development (Frugis et al. 1999). Cytokinins also seem to play a role in the induction of cyclin D transcripts, which are required for cell division (Gaudin et al. 2000; Soni et al. 1995).

Phytochrome-responsive genes have provided significant insights into the mechanisms by which phytochrome controls growth and development. Expression of GA biosynthesis genes and interactions of GA with ABA and sugar signaling indicate that phytochrome control of dormancy probably acts, at least in part, through GA and ABA (Neff et al. 2000). In support of this hypothesis, recent work has shown that phytochrome biosynthesis and ABA signaling genes map to two known QTLs for bud dormancy in poplar (Frewen et al. 2000).

With this information, the method by which environmental stimuli such as light and temperature may control dormancy induction and termination through specific plant hormones is becoming more evident. Light and temperature clearly influence production of sugar and alter phytochrome signaling. Both of these signals in turn affect ABA and GA biosynthesis. Certainly ABA, and probably GA, influences adventitious bud growth through the action of *Ick1* and possibly other cell cycle inhibitors. In addition to environmental signals, the action of auxin and cytokinins produced by growing meristems also clearly affects cell cycle activity.

Additional studies have also indicated a substantial amount of cross-talk between the signaling pathways of auxin, cytokinin, GA, ABA, and sugar/light (Chaloupkova and Smart 1994; Wingler et al. 1998; Xin et al. 1998). For example, sugar appears to be antagonistic to the signaling pathway through which cytokinin induces the expression of *Wpk4,* [a putative protein kinase in wheat (*Triticum aestivum* L.)] (Ikeda et al. 1999). Also, cytokinin signaling is coupled to the ethylene response in root and hypocotyl elongation (Cary et al. 1995). ABA may inhibit root elongation during drought by changes in ethylene levels (Spollen et al. 2000). Sugar and cytokinin have both been shown to interact with auxin signaling in control of cyclin D3 in plants (Soni et al. 1995). Additionally, sugar has been shown to be antagonistic to the GA response in several plant systems (Chao et al. 2000; Perata et al. 1997).

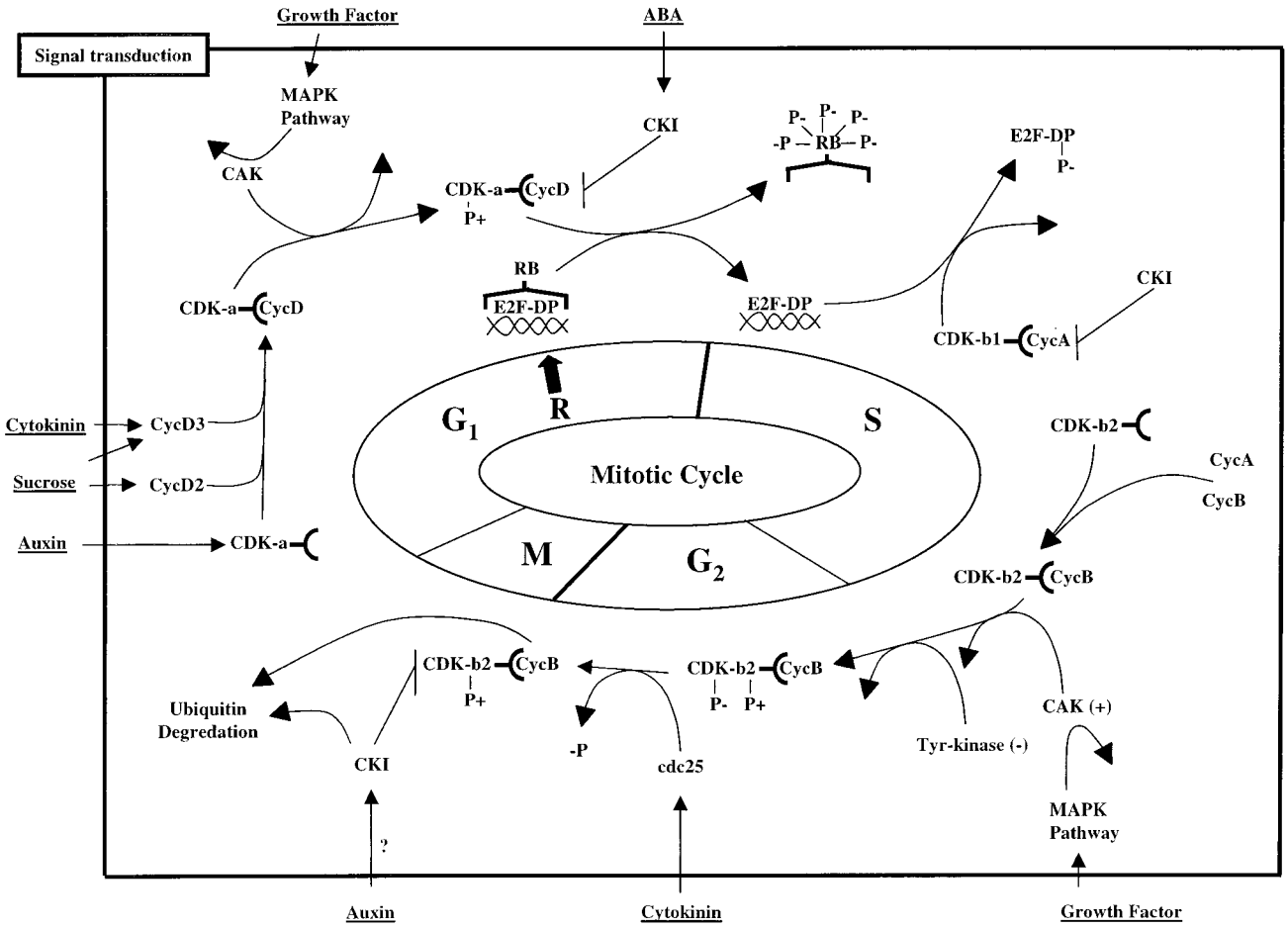

FIGURE 1. Model of plant cell cycle regulation. Based on the current information available, a simplified model for plant cell cycle regulation is proposed. Various plant hormones and growth factors initiate the up-regulation of D-type cyclins (CycD2, CycD3), which associate with a-type cyclin-dependent protein kinase (CDK) to form a CDK-a/CycD complex. CDK-a/CycD is activated by CDK-activating kinase (CAK) through phosphorylation. Active CDK-a/CycD hyperphosphorylates retinoblastoma (RB), which inhibits its binding to transcription factors like E2F, thus initiating DNA-replication and S-phase transition. The increased abundance of a-type cyclins (CycA) at S-phase probably allows CDK-b1/CycA complexes to inhibit transcriptional regulation by phosphorylating DNA-binding protein (DP). At G_2, the sysnthesis of b-type cyclins (CycB) allows for CDK-b2/CycB complex formation, which is positively phosphorylated by CAK but negatively phosphorylated by a Tyr-kinase. A cytokinin-regulated tyrosine phosphatase (cdc25) removes the inhibitory phosphate and allows for G_2/M transition. CycA and CycB contain a signal sequence that marks them for degradation through a unbiquitin proteolysis pathway. It is also evident that all the CDK complexes can be inhibited by CDK-inhibitors (CKIs), some of which are regulated through signal transduction pathways. Cell cycle phases: R = restriction point; G_1 = Gap 1; S = synthesis; G_2 = Gap 2; and M = mitosis.

Control of Dormancy at the Cell Cycle Level

As described above, growth arrest in axillary and adventitious buds can be attributed to either paradormancy (correlative inhibition and apical dominance), endodormancy (innately dormant), or ecodormancy (controlled by external environmental factors). The exact signaling mechanisms that control the onset of various phases of dormancy during bud development are not yet fully understood, but recent advances are helping us to better understand the regulation of bud growth and development at the cellular and molecular level. However, because dormancy often involves reducing the rate of cell division, some of the signaling mechanisms that control dormancy must, at some level, interact with the signaling mechanisms involved in cell division. Thus, it seems logical that understanding the signaling mechanisms that regulate cell cycle progression will provide scientists with an avenue for monitoring early events associated with the breaking of dormancy. The following section reviews known regulatory points within the cell cycle and speculates on how signaling mechanisms interact with these key regulators to control cell cycle progression.

Following each round of mitosis in a meristem, new cells either commit to a new round of DNA replication and cell division, undergo differentiation, or enter a state of quiescence, dormancy, or senescence. A commitment to reenter the cell cycle is usually regulated at the G_1-phase restriction (R) point and has been linked to protein kinase phosphorylation/dephosphorylation cascades, which are in turn activated by plasma membrane signaling machinery responsive to environmental or hormonal cues (Figure 1). In these cases, dormancy-imposed growth arrest is initiated prior to DNA replication (S-phase) and is often accompanied by reduced (but not a complete lack of) cellular activity. For example, during endodormancy, potato tuber meristems are arrested in G_1 (Campbell et al. 1996) and exhibit reduced rates of DNA, RNA, and protein syntheses (Korableva and Ladyzhenskaya 1995). In all eukaryotes, progression past the G_1/S and G_2/M mitotic checkpoints of the cell cycle is tightly regulated by a distinct class of serine/threonine pro-

tein kinases known as cyclin-dependent kinases (CDKs). CDKs were first identified as mitotic checkpoint regulators in fission yeast, in which a single gene mutation blocked DNA synthesis at G_1/S or entry into mitosis at G_2/M (Nurse and Bissett 1981). The role of phosphorylation in plant cell cycle regulation was shown through the use of kinase inhibitors, which blocked entry into mitosis and caused disassembly of the preprophase band (Katsuta and Shibaoka 1992), and by yeast two-hybrid complementation studies, which demonstrated that plant *Cdk*s could rescue cell cycle kinase mutants in yeast (Mironov et al. 1999).

In both plants and animals, CDKs are heterodimers that are composed of a catalytic subunit (the kinase) and a cyclin regulatory subunit (hence, cyclin-dependent kinase). Various combinations of CDKs control the phosphorylation status of other cell cycle regulatory components that govern the progression past the G_1 restriction point, DNA synthesis, and mitosis (Figure 1), ultimately leading to the formation of two new daughter cells (Huntley and Murray 1999; Johnson and Walker 1999; Mironov et al. 1999). However, CDKs are further regulated by CDK inhibitors (CKIs) (Wang et al. 1998) and by their own phosphorylation status. In most eukaryotes, initial commitment to enter the cell cycle appears to be highly correlated with CDKs that associate with up-regulated D-type cyclin partners. A plant CDK-activating kinase (CAK) activates CDK-a/cyclin D complexes by phosphorylating a specific threonine residue within the T-loop of the kinase subunit (Umeda et al. 1998; Yamaguchi et al. 1998a). In turn, the activated CDK-a/cyclin D complex hyperphosphorylates a retinoblastoma (RB) homologue (Nakagami et al. 1999), which releases transcription factors such as E2F (Sekine et al. 1999) that initiate transcription of E2F-regulated genes (Gutierrez 1998; Kaelin 1999). Because most cells are arrested in G_1, the growth factors (hormones) and signaling mechanisms involved in controlling expression, assembly, and activity of these gene products/complexes are likely key triggering events for release from dormancy. Further progression through the S, G_2, and M stages of the cell cycle are regulated in plants by b-type CDKs, which associate with a- and b-type cyclins. CDK-b2 contains both negative and positive phosphorylation sites that are regulated by other kinases and phosphatases. Spacial regulation and abundance of cyclin partners of CDKs are further regulated by ubiquitin-degradation pathways (Koepp et al. 1999). Thus, the commitment of quiescent and dormant cells of developing buds to either reenter or continue through the cell cycle is regulated by a highly orchestrated series of events with multiple layers of regulation, many of which are linked to signal transduction mechanisms that respond to plant growth factors (Figure 1). At present, these key genetic components serve as excellent tools for monitoring early events associated with the breaking of dormancy and, further, allow scientists to dissect the signaling mechanisms associated with their regulation.

Dormancy status in vegetative buds is likely regulated by positively or negatively regulated transcription factors that control appropriate patterns of gene expression. Multiple signal transduction pathways can be activated by either proliferative signals (e.g., growth factors) to promote growth, differentiation, and cell cycle regulation or by antiproliferative signals (stress, DNA damage, environmental factors, etc.) that can inhibit growth, differentiation, and cell cycle progression (Cook et al. 2000). In eukaryotes, signal transduction pathways work through phosphorylation/dephosphorylation cascades involving protein kinases and phosphatases, which regulate various aspects of transcription factor function, such as cellular localization, protein stability, protein–protein interactions, and DNA binding (Whitmarsh and Davis 2000). These phosphorylation signaling cascades are essential for signal transduction between growth-factor receptors and the nucleus (Pines 1999).

Mitogen-activated protein kinase (MAPK) pathways, sometimes referred to as extracellular-signal-regulated kinases (ERKs), are one of the best studied signal transduction pathways that play a central role in signaling cells to progress past the G_1/S boundary (Figure 2) (Meskiene and Hirt 2000; Roberts et al. 2000). These growth-factor signaling pathways are involved in the up-regulation of cyclin D1 and CKIs (Cook et al. 2000) and in activation of CAK (Chiariello et al. 2000). Recently, components of the MAPK signal transduction pathways have also been linked with oxidative stress-induced cell cycle arrest at G_2/M (Chien et al. 2000; Kurata 2000). Plant hormones have clearly been shown to stimulate MAPK signaling cascades (Meskiene and Hirt 2000). Thus, based on the above information, it is highly likely that the same or similar signal transduction pathways are probably involved in the regulation of dormancy in axillary and adventitious buds of perennial plants.

The way in which cellular and extracellular signal transduction mechanisms work to control the various states of dormancy in UABs of perennials such as leafy spurge is not yet understood. However, recent studies in leafy spurge have indicated that paradormant arrest of UABs probably occurs via a signal transduction mechanism that arrests cells prior to the G_1/S checkpoint (Horvath and Anderson 2000). CDK complexes immunoprecipitated from UABs of leafy spurge with a cyclin D1 antibody do show increased phosphorylation of RB 24 h after removal of the foliar tissue and over a 50% increase in the phosphorylation of RB by 48 h postdefoliation. Similarly, there is a marked increase in histone H1 phosphorylation by affinity-purified CDK complexes 24 to 48 h postdefoliation (Hovath and Anderson 2000). Also, expression of histone H3 (an indicator of S-phase transition) is up-regulated by 36 h after defoliation (Anderson and Horvath 2001). As previously stated, we are now able to monitor early events associated with cell cycle progression and, thus, the breaking of dormancy. Pinpointing these early changes allows us to focus on the genetic components of signaling mechanisms associated with these indicators.

Future Directions

In order to elucidate the basic biochemistry and signaling pathways occurring during bud arrest, cloning of genes involved in this process undoubtedly is the foremost means to unravel these questions. Recent advances in plant genetics and genomics have offered unprecedented opportunities for identifying differentially expressed genes/proteins and gene function. The function of genes responsible for specific phenotypes such as early dormancy, late dormancy, or loss of dormancy may be investigated by initiating QTL analysis (Frewen et al. 2000; Paterson et al. 1995), performing gene traps (Springer 2000), developing genetic maps, analyzing

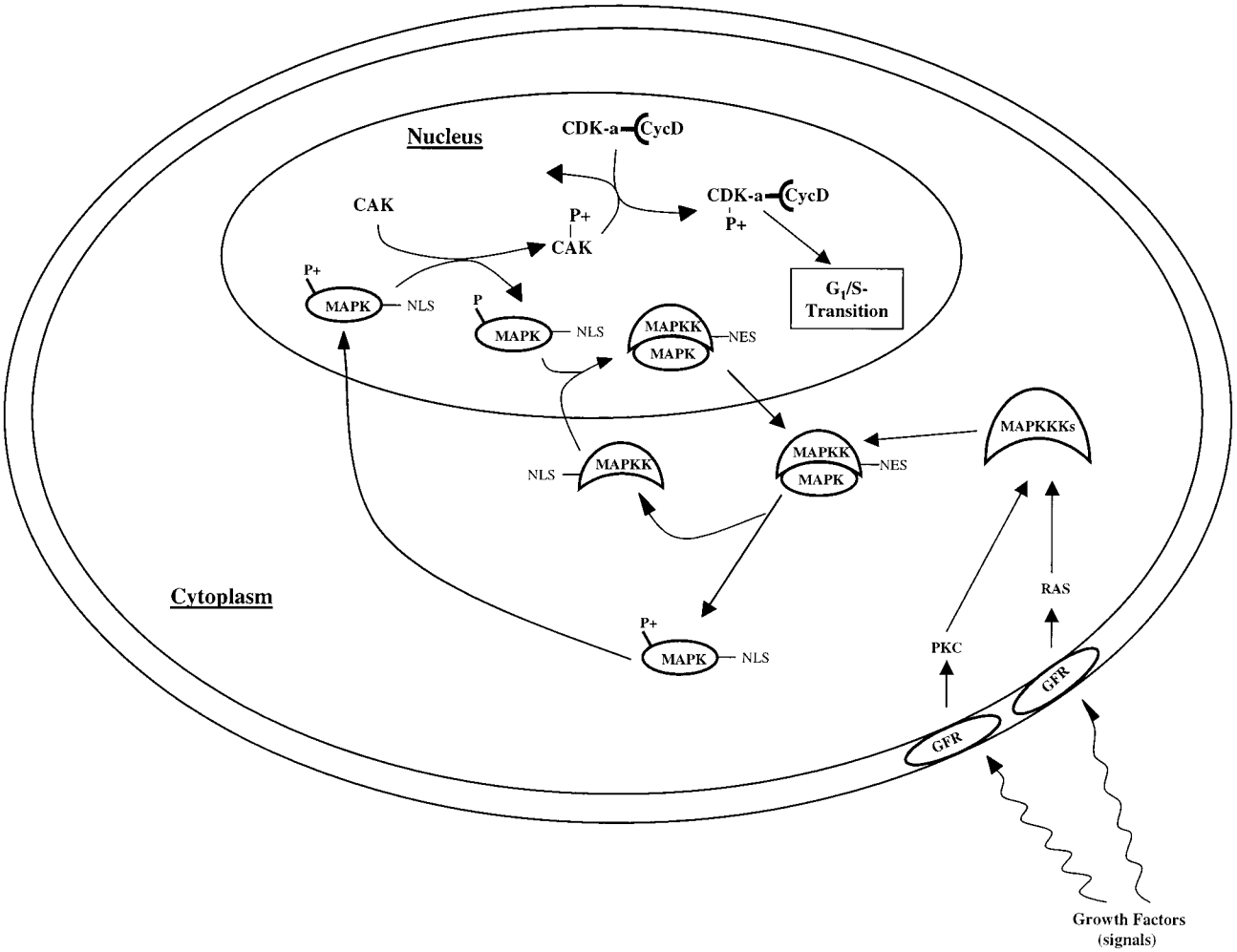

FIGURE 2. Model of signal transduction pathway controlling G_1/S transition. Based on information available from eukaryotes, we propose the following model for a signal transduction system for the plant G_1/S cell cycle transition. Proliferative growth factors (possibly plant hormones) bind to plasma membrane receptors, which activate a mitogen-activated protein kinase (MAPK) cascade. Activation of MAPKKK regulates MAPKK activation, which in turn activates MAPK. This pathway controls signaling from the receptor to the nucleus, where MAPK activates cyclin-dependent protein kinase (CDK)-activating kinase (CAK), which in turn, activates CDK-a/CycD, and the subsequent phosphorylation of retinoblastoma intiates the G_1/S transition. Protein kinase C, PKC; RAS, GTPase; nuclear export signal, NES; nuclear localization signal, NLS; growth factor receptor, GFR.

gene knockout mutants (Krysan et al. 1999), cluster analysis of coordinately expressed genes (Eisen et al. 1998; Wen et al. 1998), and other mechanisms. Changes in gene or protein expression in response to internal or external signals involved in the regulation of dormancy may be studied using various methods, including two-dimensional polyacrylamide gel electrophoresis (Dutt and Lee 2000), differential display (Liang and Pardee 1992), serial analysis of gene expression (Velculescu et al. 1995), and microarray (Aharoni et al. 2000; Reymond et al. 2000; van Hal et al. 2000).

The microarray technology has been developed and widely used for investigating diverse problems in both plants and animals in the last 5 yr. Because it has the capability to simultaneously detect and quantitate a large number of differences in gene expression related to pathways and mechanisms, it is an ideal method for studying complex phenomenon controlled by several interacting signaling pathways. This technology has been used in identifying genes of specific functions (Aharoni et al. 2000), comparing transcript profiles under different environmental conditions (Reymond et al. 2000), evaluating transcript profiles between genetically modified and control species (van Hal et al. 2000), and characterizing differentially expressed genes between tumor and normal cells (Epstein and Butow 2000). We also should be able to develop a better understanding of dormancy regulation in UABs of leafy spurge through DNA microarray analysis. Early analysis of ESTs from growth-induced leafy spurge UABs indicates expression of numerous cell cycle regulatory and signal transduction genes encoding proteins such as receptor-like protein kinases, serine/threonine protein kinases, RAB-like GTPase activator proteins, GTP-binding proteins, MAPK, MAPKK, RB, CAK, cyclin-selective ubiquitin, histones H1, H2A, H2B, and H3, histone acetyltransferase, 14-3-3, tubulin, and DNA-binding transcription factors (www.ncbi.nlm.nih.gov, keyword: Euphorbia esula). The existence of other leafy spurge ESTs responsive to ABA, GA, cytokinin, and ethylene should enhance our ability to further address factors associated with the signal transduction mechanisms that regulate the various types of dormancy in axillary and adventitious buds of vegetative propagules. Information gained from these new studies should allow scientists to improve future weed management strategies. For example, understanding/identifying specific genetic components of the cell cycle machinery will

allow development of designer herbicides to block critical steps associated with signal transduction pathways.

Obviously, new knowledge will be a key to improving the effectiveness of existing control measures and to developing alternative control strategies. The progress in molecular mechanisms may allow us to explore and develop novel ways to kill or manage perennial weeds. A promising example is to use a virus to control weed growth based on the results of virus-induced gene silencing (VIGS) in plants (Burton et al. 2000; Kjemtrup et al. 1998; Kumagai et al. 1995; Ruiz et al. 1998). It is now known that a virus vector carrying host gene fragments may prevent expression of homologous, chromosomal genes of the host. VIGS can be used for quick functional analysis of unknown genes. At least three different virus vectors have been used for function studies in tobacco (*Nicotiana*) species (Burton et al. 2000; Kjemtrup et al. 1998; Kumagai et al. 1995; Ruiz et al. 1998). This technology can be extended to introduce a gene required for growth into the host plant via virus and to inhibit the host's ability to grow. The merit of this approach is that it is extremely specific, because only those plants carrying genes highly similar (80% in sequence identity or higher) to those in the engineered viruses should be affected (Ruiz et al. 1998). Besides, a virus can propagate itself and systemically spread from tissue to tissue, avoiding the need for repeated application. However, such a virus can also be engineered to limit its propagation and, thus, to provide the farmer with very specific and deadly herbicides. The use of VIGS also allows for high throughput screening of genes identified from microarray analysis and thus should be a powerful tool for determination of genetic components important in the regulation of bud dormancy.

Summary

This review covers old and new aspects on the physiology, molecular biology, and genetics of bud dormancy. Because dormancy is linked to growth arrest, we suggest the importance of understanding signal transduction mechanisms involved in controlling cell division and differentiation. We now know that multiple growth factors are involved in control of bud dormancy status. However, further knowledge is needed to fully understand how environment and plant growth regulators work together to affect whole-plant physiology to impose bud dormancy. Many molecular techniques are currently available for identifying genetic components involved in the signaling mechanisms regulating bud dormancy. DNA microarray is an important technological advancement that will allow quick screening for genes that are critical to the regulation of bud dormancy. Further use of VIGS technology should allow for functional analysis of important genetic components correlated with dormancy and dormancy-breaking events. Such information could lead to new weed management strategies for perennial weeds, such as the development of DNA-based herbicides.

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